Postoperative chylothorax successfully treated using conservative strategies

Victoria Stager, MD, Lam Le, MD, and Richard E. Wood, MD

The anatomy of the thoracic duct varies considerably, rendering it prone to disruption during thoracic surgery. Chylothorax complicates up to 0.5% of all intrathoracic procedures, its morbidity requiring the surgeon's vigilance throughout the entire course of the patient's illness. The natural history of chylothorax includes cardiopulmonary dysfunction, immunosuppression, nutritional and electrolyte derangements, and, ultimately, sepsis and death. General criteria for conservative management of thoracic duct injuries have been described in surgical literature, yet in selecting the best treatment for the patient, surgeons rely heavily on their own experience and the patient's unique presentation. A variety of conservative strategies may be implemented with success in approximately 50% of all cases. The duration of conservative treatment may vary, but the futility of conservative efforts should be recognized early and surgical intervention not delayed. We report a case of a 42-year-old man who presented at our institution with chylothorax after posterior mediastinal mass resection. The patient was treated successfully by withholding oral food and fluids, instituting total parenteral nutrition, and draining with a thoracostomy tube. He was discharged home with a complete resolution of chylothorax on hospital day 8. We describe the patient's illness course and discuss current strategies in the conservative management of thoracic duct injury after mediastinal resection.

CASE PRESENTATION

A 42-year-old man who recently underwent a resection of a benign posterior mediastinal mass came for an office visit concerned with increasing shortness of breath and chest pain since his surgery 7 days prior. Additionally, he complained of fatigue, decreased appetite, and exercise intolerance. He denied palpitations, cough, dysphagia, or fever. His blood pressure was 130/88 mm Hg; heart rate, 80 beats per minute; respiratory rate, 20 respirations per minute; and oxygen saturation, 90% on room air. His temperature was 36.3°C. On physical examination, the patient was a pleasant man who looked his stated age but appeared tired. He had normal heart sounds and decreased breath sounds of the right chest. He did not have jugular venous distention. His thoracotomy incision had healed well. The rest of the examination was unremarkable. The patient's past medical history included recent surgery as described below, pancreatitis, hypertension, anxiety, and alcohol abuse.

A week before this visit, the patient underwent an open resection of a benign mass in the posterior mediastinum of the right hemithorax. The mass was located in the azygoesophageal recess, extending from immediately subcarinal to the supradiaphragmatic area, and was excised via a standard right thoracotomy approach. The mass measured approximately 140 cm^3 and appeared abnormally vascular, complicating the dissection by small vessel bleeding. No other adverse findings or events were noted during the operation. Pathology subsequently identified the mass as atypical lymphoid hyperplasia, or Castleman's disease. The patient recovered appropriately and was discharged home in good condition.

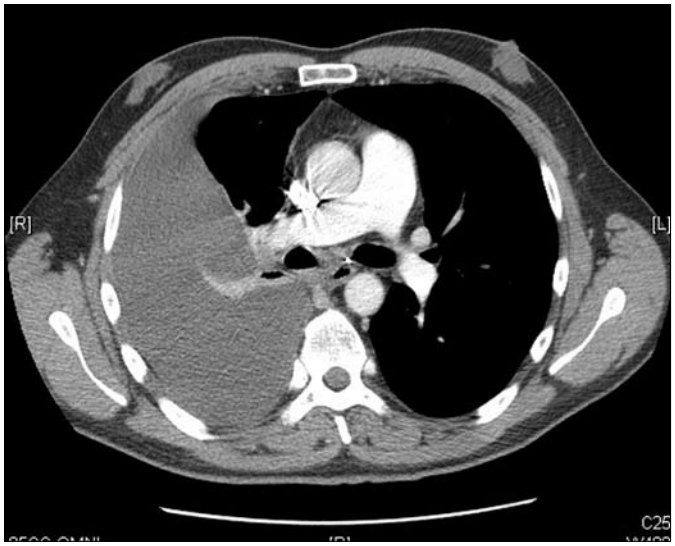

Figure 1. Chest CT obtained at the postoperative clinic visit. A large pleural effusion is present in the right hemithorax with evidence of pulmonary atelectasis and compression of the lung towards the mediastinum. The effusion is not loculated.

At his postoperative clinic visit, the patient underwent a diagnostic workup by analysis of blood count, chemistry, culture, and a chest radiograph. A computed tomography (CT) scan was performed to characterize the pathological process with greater precision. Noteworthy laboratory findings were a

From the Department of Surgery, Baylor University Medical Center, Dallas, Texas.

Corresponding author: Victoria Stager, MD, Resident, Department of Surgery, Baylor University Medical Center, 3500 Gaston Avenue, Dallas, Texas 75246 (e-mail: Victoria.Stager@BaylorHealth.edu).

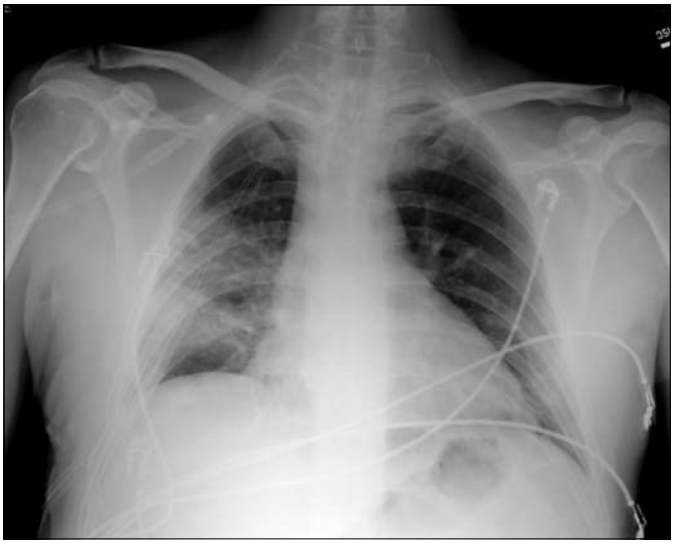

Figure 2. Posteroanterior chest radiograph on hospital day 1. The right pleural effusion has been drained with an intercostal chest tube, and an infiltrate is present in the right lower lobe.

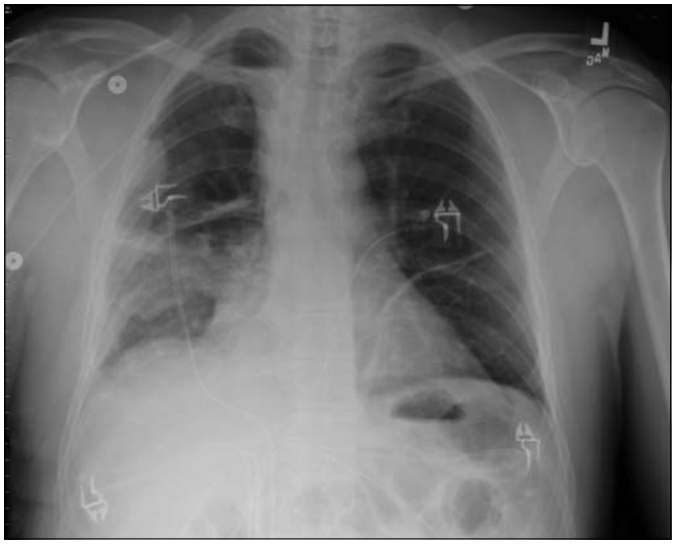

Figure 3. Posteroanterior chest radiograph following removal of the chest tube on hospital day 7. There is an infiltrate of the right lower lobe and no evidence of pleural effusion.

white blood cell count of 7000/mm^3; hemoglobin, 11.2 g/dL; hematocrit, 34.5%; albumin, 3.1 g/dL; total protein, 5.9 g/dL; and magnesium, 1.7 mg/dL. The chest radiograph showed a large pleural effusion in the right hemithorax. On CT of the chest, the effusion appeared homogenous, without loculations or pleural thickening, the size of the effusion resulting in significant atelectasis of the lung *(Figure 1).* A 28Fr chest tube was placed using a standard technique, and 3 L of milky fluid was drained immediately. A follow-up chest radiograph confirmed a complete evacuation of the effusion and a full expansion of the lung *(Figure 2).* The pleural fluid was sent for culture and a triglyceride level. The results showed no bacterial growth and a fluid triglyceride level of 3032 mg/dL, confirming the diagnosis of chylothorax.

We treated the patient with a short course of aggressive conservative therapies. A low-fat diet was initiated on admission, and orders for nothing by mouth and total parenteral nutrition were begun on hospital day 2, immediately following the formal diagnosis. The patient's electrolytes, total blood count, and intake and output were monitored daily. Chest tube output was recorded every 8 hours. In the first 24 hours, the chest tube output held at 200 mL, and the fluid appeared less milky. The next day, output decreased to 35 mL and remained <50 mL per day for the next 2 days. On day 5, as the patient was challenged with a regular oral diet, the output from the tube peaked at 112 mL and then remained negligible. The tube was removed on hospital day 7, and the follow-up chest radiograph showed no evidence of fluid *(Figure 3).* The patient went home on hospital day 8. He has since been seen in the clinic, with no sign of recurrence.

DISCUSSION

Anatomical and physiological considerations

In 1651, Parisian medical student Jean Pecquet challenged the world of science with his theory that food turned into blood not in the liver, as was presumed by his scholarly community, but in the heart, as a result of passing from lacteal vessels of the intestines to a thin-walled receptacle in the upper abdomen and then to the subclavian vein via a long, delicate tube (1, 2). Decades would pass before medical textbooks adopted this new knowledge.

The thoracic duct originates at the body of L2 from a triangular dilatation called cisterna chyli, passes posterior to the median arcuate ligament through the aortic hiatus of the diaphragm, and then travels in the posterior mediastinum of the right hemithorax, just anterior to the vertebral column, between the azygous vein and the aorta *(Figure 4).* At the level of the fourth to sixth thoracic vertebrae, it crosses over to the left hemithorax and ascends posterior to the aortic arch, exiting the thorax through the superior thoracic aperture in the neck. It then arches anterior to the scalenus anterior muscle at the level of C7 and dives downwards, terminating at the junction of the left subclavian and internal jugular veins (3, 4).

Omitting few details, this simplified pattern of the thoracic duct's course is present in 40% to 65% of the population, belying its embryological evolution from a bilateral plexiform structure (3–6). The remainder of the population is noted to have significant variations, with the thoracic duct duplicating, triplicating, coalescing, branching out repeatedly, and then terminating either independently or as one duct. It has been noted, ironically, that the only thing constant in the anatomy of the thoracic duct is its inherent variation (3).

The anatomical unpredictability renders the thoracic duct prone to injury, even in the hands of an experienced surgeon. Iatrogenic chylothorax complicates 0.25% to 0.50% of all intrathoracic cases and has been described in association with neck dissection; cardiac surgery; resection of the lung, esophagus, or mediastinal masses; and even after placement of a central venous line (4, 5, 7). Esophagectomy is estimated to result in disruption of the thoracic duct more often, with the most frequently reported incidence around 3% to 4% (5–7).

The thoracic duct transports sterile, bacteriostatic, and slightly alkaline chyle that includes almost 70% of ingested

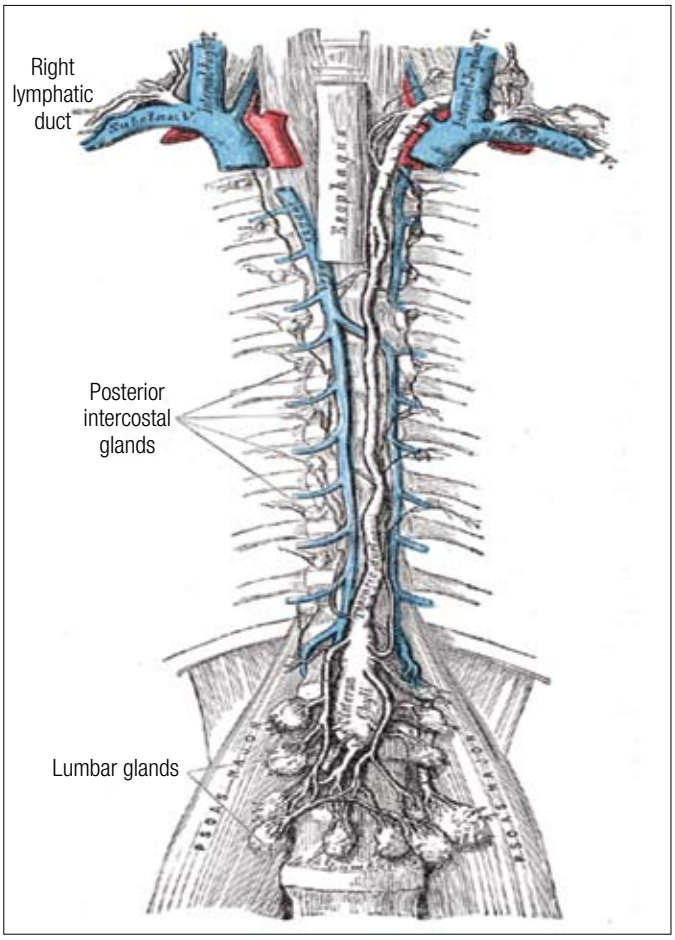

Figure 4. Anatomy of the thoracic duct. From Gray H. *Anatomy of the Human Body,* 1918.

dietary fats in the form of chylomicrons, cholesterol, and triglycerides. In contrast to long-chain fatty acids that make up the core of chylomicrons, fatty acids containing <10 carbon atoms in the chain are absorbed directly into the portal vein and are not constituents of chyle. The other components of chyle include lymphocytes (mainly T cells), erythrocytes, immunoglobulins, sugar, enzymes, fat-soluble vitamins, and extravasated plasma proteins (3). Chyle biochemistry studies show the presence of fibrinogen, antithrombin, and prothrombin (3, 7).

The morbidity of an unrecognized chyle leak results from local, immunologic, and metabolic effects, and the mortality of untreated chylothorax is estimated at 50% (3, 5, 6). Untreated chylothorax compresses the ipsilateral lung and may lead to a mediastinal shift compromising cardiopulmonary function (5, 6). Both cell-mediated and humoral mechanisms of immunity are impaired due to the loss of lymphocytes and immunoglobulins. Metabolic derangements ensue from rapid loss of albumin, fat, vitamins, and electrolytes, homeostasis being further disrupted by profound fluid shifts, hypovolemia, and metabolic acidosis (2, 5, 6, 8).

Diagnosis and initial management of chylothorax

Usually, postoperative chylothorax becomes symptomatic 2 to 10 days after the procedure. Patients frequently complain of shortness of breath and coughing. Pleuritic chest pain and fever are unusual because chyle is sterile and considered nonirritating to the pleural surface (7, 9). Chylous bronchorrhea has been described but is very rare (8). The patient's history and physical exam are followed by a chest radiograph with a lateral view. Should a pleural effusion be seen, the next diagnostic and therapeutic step in management is to drain the effusion and perform a biochemical or quantitative analysis of the pleural fluid. The milky appearance of the fluid is not a consistent property of chyle and may confuse the diagnosis, as chylothorax may present with green, serous, or bloody effusion (7).

Currently, the exact diagnosis of chylothorax is established by the presence of chylomicrons in the lipoprotein analysis of the pleural fluid (5, 7, 8, 10). This test is diagnostic because the thoracic duct is the only carrier of chylomicrons immediately after eating. It takes several hours before chylomicrons can be detected in the blood, their concentration peaking 3 hours after a meal (5). Nevertheless, a more commonly encountered method of diagnosis is the biochemical analysis of the pleural fluid. A triglyceride level of ≥110 mg/dL establishes the correct diagnosis in 99% of cases (5, 7). High lymphocyte counts (400–7000/mL) and low cholesterol (<200 mg/dL) further support the diagnosis (4). Lipoprotein analysis remains the confirmatory diagnostic test used in equivocal cases. A positive Sudan III fat stain, mentioned as a diagnostic tool in several reports, of itself is not diagnostic of chylothorax (11). The stain demonstrates different types of fatty globules, not chylomicrons specifically. In addition, quantitative criteria have not been established for diagnosis of chylothorax based on this type of cytological preparation (5, 7).

The management of postsurgical chylothorax is guided by a set of principles rather than a strict algorithm. These principles are efficient drainage of the effusion, cessation of flow through the thoracic duct, and obliteration of the pleural space (8). Current modalities include conservative, surgical, and radiation therapies. Although in some situations immediate surgery may be indicated, a short course of aggressive conservative therapy is advocated initially in most cases and is estimated to result in permanent resolution of chylothorax in 50% of patients (2, 5) (see *Figure 5*).

Placement of an intercostal tube allows complete drainage of the effusion, which leads to symptomatic relief and reexpansion of the lung. The chest tube stays at water seal, its output recorded every 8 to 24 hours (9). In a postesophagectomy patient, Wemuss-Holden advocates hourly monitoring of the chest tube output, as the mortality of conservatively treated chylothorax in these patients may reach 80% compared with 10% to 16% mortality associated with operative intervention (6, 11). Chyle production is slowed by several dietary interventions implemented as soon as the diagnosis is suspected. Some authors advocate a diet consisting of medium-chain triglycerides, which will be absorbed by the portal system, reducing the net flow through the duct (8, 12, 13). A more decisive approach is to cease all enteral intake while providing central hyperalimentation. Several studies support use of somatostatin, etilefrine, and octreotide as important adjuvant treatments among the conservative strategies (4, 5, 7, 12). Somatostatin and its analogues

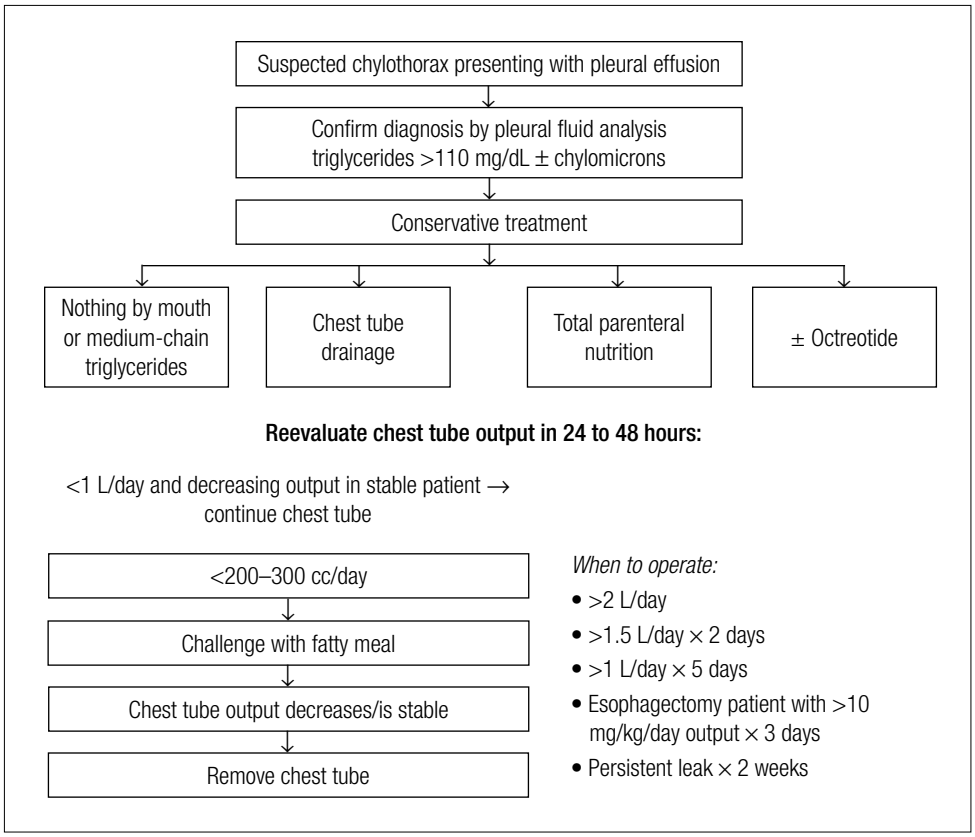

Figure 5. Approach to management of chylothorax.

have been shown to reduce chyle production, presumably by vasoconstricting the splanchnic vessels (4, 5, 7).

Of interest, early literature emphasized the importance of bed rest and elevation of the patient's head. In particular, it was specifically recommended that during expectant management, "all practical efforts should be made to diminish such activities as coughing, straining, and laughing" (9). Though not included in current literature, the advice is valid, as increased intraabdominal pressure compresses the cisterna chyli, leading to increased flow through the duct (2).

Timing of surgical intervention

The trend in the daily output from the chest tube is probably the single most important indicator of the patient's chance of success with conservative therapy. Considering that in a healthy adult, the thoracic duct transports 1.5 to 4 L of chyle daily, the amount of drainage may vary significantly in different patients, and in general, a volume ≥1000 mL/day defines a high-output leak (2, 5, 6, 8). Early recommendations frequently allowed a 2-week course of conservative treatment before surgical intervention for all patients with iatrogenic chylothorax (3, 9, 14, 15). In contrast, current literature differentiates the length of the conservative trial based on the specific cause of chylothorax. Most authors agree that postesophagectomy patients should undergo early operation (in <5 days) (6, 16). Wemyss-Holden et al in 2001 reviewed postesophagectomy chylothorax and concluded that conservative therapy with vigilant monitoring of chylous output and metabolic parameters for 48 to 72 hours after the diagnosis is most appropriate. After this period of time, if drainage remains >10 mL/kg/day, surgical intervention is indicated (6). Other recent studies evaluate management of chylothorax arising from various thoracic procedures, including mediastinal, lung, heart, and esophageal surgery (besides esophagectomy for cancer), and conclude that spontaneous resolution of chylothorax is unlikely with a fluid loss of 1.5 L/day for 1 to 2 days, or >1 L/day for 5 days (2, 5, 8). In fact, output >1 L on day 5 is considered to be a negative prognostic factor (5). In addition, if the fluid losses reach 2 L/day, repeated or continued drainage is contraindicated (8).

There is no consensus regarding resumption of the oral diet. According to a review by Cerfolio in 2006 (11), the oral diet should begin when the output from the chest tube is <200 to 300 mL/day, but this recommendation is not consistently found in the literature. Cerfolio recommends challenging the lymphatic system with a fatty meal instead of a slow progression from a low-fat to regular diet (11). If, while on the high-fat diet, the output remains nonchylous and low for the next 2 days, the chest tube may be removed (11). There are no formal guidelines regarding the amount of drainage favorable for the removal of the chest tube.

In the case of our patient, suspicion of an injury to the thoracic duct was high. The insidious worsening of compression-related symptoms, absence of fever/infection, and a pleural effusion in the setting of a recent thoracic procedure suggested the clinical diagnosis. The inadvertent bleeding in the area of dissection and shrinking of the thoracic duct due to preoperative fasting precluded visualization of the thoracic duct intraoperatively. Assuming the patient's thoracic duct anatomy followed a near-standard pattern, the area of dissection below T4 predictably resulted in a right rather than left chylothorax.

In management of this patient, we intended to limit the duration of conservative strategies to 4 to 5 days if no improvement resulted. However, a significant decline in the amount of pleural drainage occurred surprisingly soon, raising a question: When did the closure of the leaking area occur? Since the thoracic duct was not visualized at the time of surgery, the location of the chylous leak can only be hypothesized. In a healthy individual, the visceral and the parietal pleura are separated by a thin layer of pleural fluid and are not in direct contact. However, when the parietal pleural surface is disrupted, the inflammatory reaction is likely to cause adhesions between the visceral pleura of the lung and the area of dissection. In our patient, the thoracic duct or its disrupted tributary may have closed spontaneously

around the time of admission, postoperative adhesive processes in the surrounding tissue facilitating the closure. The reexpansion of the lung after the chest tube placement may have further obliterated the pleural space compressing the injured area. Alternatively, had the injury been limited to a small tributary of the thoracic duct, cessation of chyle flow by dietary changes could have resulted in collapse and closure of the thin-walled lymphatic conduit.

CONCLUSIONS

Anomalies of the thoracic duct anatomy are almost as common as the usual pattern. This anatomical variation of the thoracic duct is of surgical significance, as iatrogenic chylothorax complicates 0.25% to 0.50% of all intrathoracic operations, with mediastinal dissection or esophagectomy cited as most common. Timely recognition of the injury is critical, as the loss of chyle may rapidly lead to respiratory insufficiency, immunosuppression, and recalcitrant metabolic derangements. Iatrogenic chylothorax arising from esophagectomy necessitates a very short, 1- to 3-day conservative trial, while other circumstances of injury may allow continuation of nonoperative treatment for several more days depending on the drainage amount and the patient's condition. A 5-day course of conservative strategies may be implemented in most situations and should include thoracostomy drainage, avoidance of oral intake, fluids, and hyperalimentation. Importantly, the hemodynamic, metabolic, and nutritional aspects of the patient's care should be approached aggressively due to the rapidly deteriorating nature of the condition. Somatostatin/octreotide may be implemented as an additional therapy. If, despite aggressive medical management of the injury, significant fluid loss continues for 5 to 7 days, surgical intervention is indicated.

1. Lewis SJ. *Jean Pecquet (1622–1674) and the Thoracic Duct.* PhD thesis, Yale University, 2003.
2. Merrigan BA, Winter DC, O'Sullivan GC. Chylothorax. *Br J Surg* 1997; 84(1):15–20.
3. Miller JI. Anatomy of the thoracic duct and chylothorax. In Shields TW, Locicero J, Ponn RB, Rusch VW, eds. *General Thoracic Surgery,* 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2005:887–889.
4. Rodgers BM. The thoracic duct and the management of chylothorax. In Kaiser LR, Kron IK, Spary TL, eds. *Mastery of Cardiothoracic Surgery,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2006:244–253.
5. McGrath EE, Blades Z, Anderson PB. Chylothorax: Aetiology, diagnosis and therapeutic options. *Respir Med* 2009 Sep 17 [Epub ahead of print].
6. Wemyss-Holden SA, Launois B, Maddern GJ. Management of thoracic duct injuries after oesophagectomy. *Br J Surg* 2001;88(11):1442–1448.
7. Nair SK, Petko M, Hayward MP. Aetiology and management of chylothorax in adults. *Eur J Cardiothorac Surg* 2007;32(2):362–369.
8. Browse NL, Allen DR, Wilson NM. Management of chylothorax. *Br J Surg* 1997;84(12):1711–1716.
9. Williams KR, Burford TH. The management of chylothorax. *Ann Surg* 1964;160:131–140.
10. Agrawal V, Doelken P, Sahn SA. Pleural fluid analysis in chylous pleural effusion. *Chest* 2008;133(6):1436–1441.
11. Cerfolio RJ. Chylothorax after esophagogastrectomy. *Thorac Surg Clin* 2006;16(1):49–52.
12. Leibovitch I, Mor Y, Golomb J, Ramon J. The diagnosis and management of postoperative chylous ascites. *J Urol* 2002;167(2 Pt 1):449–457.
13. Su IC, Chen CM. Spontaneous healing of retroperitoneal chylous leakage following anterior lumbar spinal surgery: a case report and literature review. *Eur Spine J* 2007;16(Suppl 3):332–337.
14. Connolly JE, Smith JW. The management of chylothorax. *Calif Med* 1959;91(2):75–78.
15. Selle JG, Snyder WH 3rd, Schreiber JT. Chylothorax: indications for surgery. *Ann Surg* 1973;177(2):245–249.
16. Merigliano S, Molena D, Ruol A, Zaninotto G, Cagol M, Scappin S, Ancona E. Chylothorax complicating esophagectomy for cancer: a plea for early thoracic duct ligation. *J Thorac Cardiovasc Surg* 2000;119(3):453–457.